

ENCAPSULATED HYDROTHORAX (HYDROTHORAX SACCATUS INTERLOBARIS) IN ASSOCIATION WITH MYOCARDIAL INSUFFICIENCY.

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The encapsulation of fluid between the layers of the pleura develops frequently either as a localized extravasation or as the result of the walling off of a part of a general one. Ordinarily, when serous fluid collects in the pleural space, it is free, and circumscribed pockets are not formed. If sacculation of a transudate or of a non-purulent exudate develops, it may be, as Gerhardt suggested, because an antecedent pleurisy has led to the formation of local adhesions that segregate portions of the thoracic cavity into which the fluid exudes; as Ottner and others believe that adhesions develop at the onset of a pleurisy and form boundaries for fluid extravasated subsequently; or that the sacculation represents the local remains of a general exudate or transudate, most of which had been reabsorbed.

Such localized collections of fluid in the pleural space can be recognized when the physical signs of fluid are found in a limited portion of the chest, but the detection and the accurate localization of them has been made more readily since the advent of the roentgenograph. Utilization of the latter has made it possible clinically to demonstrate that the accumulation between the parietal and visceral pleura may be limited to an area in contact with the lateral, the anterior or the posterior thoracic walls, between the diaphragm and the inferior surface of the lungs, between the superior surface of the thorax and the pulmonary apex, between the mediastinum and the medial margin of the lung, or that it may lie entirely within an interlobar fissure.

The physical signs of a circumscribed collection of fluid within a septum between the lobes of a lung may be so slight that they defy detection. The demonstration of a localized area of flatness over which there is suppression of the respiratory murmur and of vocal resonance, suspended in the anatomical region of a fissure and the

roentgenographic demonstration there of a dense shadow surrounded by normal pulmonary fields is almost pathognomonic of fluid limited to an interlobar fissure. The location of such a shadow will be determined by the site and the course of the fissure and the portion of it occupied by the collection of fluid. The size and shape of it will vary according to the amount of the fluid and the degree of compression of the contiguous lung. The long axis of the shadow will be from the hilum toward the lateral wall of the thorax in a more or less oblique direction. Ottner¹ states that interlobar effusions are separated from the hilum by a band of air—containing lung, but Assman² and Fleishchner³ assert that this finding is not present, except in those cases in which the median portion of the fissure has been obliterated by adhesions. Dependent upon the direction in which the Roentgen rays are projected, the shadows of such interlobar collections of fluid may be spindle or band shaped with sharply demarcated margins on all sides. However, if the fluid does not fill all of the fissure or if it is a large collection and compresses the adjacent lung, this typical contour may be lacking and the film will show a round, oval or ovoid area of increased density. In lateral or in oblique views of the chest, the shadows may be ribbon or wedge shape and have linear or curved outlines, but when roentgenographs made thus are viewed, the borders of the abnormal shadows are sharply defined unless some disease of the neighboring lung, marked thickening of the pleura or a large effusion between the parietal and visceral pleura obscure them.

Similar roentgenographic appearances in films made by the antero-posterior projection of the X-ray are seen occasionally when there is a marginal pneumonia about the fissure or an area of pulmonary excavation near it, but although such shadows may have one clearly defined edge, generally the others are irregular and hazy. At times pleural cysts present difficulties of diagnosis that are insurmountable unless all available data are collated. The shadows of malignant neoplasms of the lung can be recognized if there are evidences of invasion of the pulmonary parenchyma, and tumors of the bronchi can be identified by bronchoscopic examination or by roentgenographic observation after the introduction of lipiodol into the bronchial arborizations. The Roentgen ray affords the maximum assistance only if it is projected from several different angles and

of these the lateral projection or in the Kreuzhohlstellung are the most informative.

A correlation of the history of the patient with the data acquired by physical examination, by bacteriological and roentgenographic study has identified as the causes of such localized exudates: (1) Tuberculosis; (2) Meta and parapneumonic infections; (3) Penetrating wounds of the thorax; and (4) Metastatic infections transported through the vascular system (tonsillitis, scarlet fever, puerperal sepsis, etc.) or via the lymphatics (peritonitis, sub-phrenic abscess).

Although Laennec described an instance of hydrothorax in which the accumulation of the fluid was limited by an antecedent obliterative pleurisy to the lower half of the right thoracic cavity in a patient who had died of myocardial disease, and although some clinicians have asserted that a pleural transudate may be loculated, many observers maintain and Anders, as recently as 1913, stated that "serous collections are never encapsulated."⁴ It seems strange that so few instances of localized hydrothorax or "hydrothorax saccatus" have been recorded. This may be due in part to the fact that although pleural adhesions were present before the serous extravasation occurred, they caused complete obliteration of the potential pleural space, or it may be that fibrous bands divided it into areas so large that loculation was not considered. *A priori*, it would seem that whenever local or general vascular stasis or a state of lowered colloid osmotic pressure of the blood leads to hydrothorax in an individual who has preformed bands of pleural adhesions, a sacculatation of the transudate would result, but although theoretically these physical conditions should perhaps have been encountered now and again in the several morbid states in which hydrothorax occurs (mediastinal tumors, myocardial failure, nephritis, nephrosis, leukemia, severe anemias, etc.) this finding has been reported only rarely in patients who had cardiac disease.

In 1926, Fleischner⁵ reported the roentgenographic detection of a round, sharply demarcated shadow in the right thorax of a patient with aortic insufficiency and dilatation and calcification of the aorta that varied from time to time while he was under observation and that autopsy proved to be fluid encapsulated between the upper and

middle lobes of the lung. He cited a similar case that had been described by Helm.

Some years later, Stewart⁵ recorded another instance of the same sort. His patient was a woman aged 64 years whose thoracic roentgenograph showed, during the time she had congestive cardiac failure a sharply outlined shadow 5.3 x 5 cm. in the right interlobar fissure. This shadow remained unaltered for four or five days of treatment during which the myocardial condition improved, but it diminished in size and disappeared within two months after the occurrence of a febrile illness associated with the occlusion of a coronary artery. Two weeks after recovery from this syndrome and the resumption of physical activity symptoms of circulatory embarrassment recurred and the shadow returned, to disappear once more after treatment had been carried out for four weeks. A like recurrence and disappearance of symptoms and of the shadow were noted again after an interval of two months and for a fourth time eight months later, shortly before the death of the patient. Post-mortem examination showed calcification and stenosis of the aortic valves; malformation of the aortic valves; cardiac hypertrophy; advanced atherosclerosis of the aorta; moderate atheromatosis of the aortic cusp of the mitral valve and of the coronary vessels; atherosclerosis and infarcts of the kidneys; ascites; edema of the legs and intestines; serous pericarditis; serofibrinous pleurisy of the left cavity with few old adhesions; obliteration of the right pleural cavity with the exception of a small space between the upper and middle lobes, which was filled with serofibrinous fluid; pleural induration over both apices; osteoporosis of the sternum.

E. P. Kiser⁶ observed a man 51 years of age with congestive myocardial failure and hypertension in whom a "shadow was seen in the right chest in the region of the right interlobar space. It was distinctly ovoid in shape and extended 8 cm. to the left of the right thoracic wall and was 5 cm. in width. In density the shadow was identical with that of the pericardial effusion that was present." Approximately six weeks later when cardiac compensation had been re-established the pericardial effusion had disappeared, the blood pressure was normal and another film showed no interlobar pleural abnormality.

E. Freedman⁷ reported the presence of a "well-demarcated circular shadow of increased density, 6 x 7 cm. in diameter, near the upper border of the hilum of the right lung" in a man 76 years of age that autopsy proved to be due to a localized effusion of 60 c.c. of clear fluid in the fissure between the upper and the middle lobe of the right lung. The patient had also an occlusion of the coronary artery, an aneurysm of the posterior wall of the left ventricle and a rupture of the bladder. Steele⁸ has recorded two similar cases.

The paucity of published reports of encapsulated hydrothorax in cases of myocardial failure and the general lack of knowledge concerning the occurrence of the condition warrant the narration in detail of the following record.

T. C., an Italian street vendor aged 58, was admitted to the Sinai Hospital* for the first time on December 8th, 1929, complaining of "shortness of breath, swelling of the feet and thumping of the heart." His family history was non-essential. He stated that he had had "rheumatism" in 1895 and a specific urethritis in 1910. An attack of pneumonia in 1922 was his only severe respiratory infection and he had never had a chronic cough, hemoptysis, pleural pain or an unexplained febrile illness. He denied syphilitic infection by name and by symptom.

Until 1925 he enjoyed good health, but in that year dyspnea on exertion developed. In November, 1929, this symptom became so much more severe that it was necessary for him frequently to stand or to crouch in order to breathe. Such paroxysms of respiratory difficulty occurred at first from two to five times daily and lasted for from two to ten minutes, accompanied at times by precordial oppression and by an unproductive cough. The attacks gradually became more severe, edema of the feet developed and on December 3rd, general discomfort was so marked that he came to the hospital for relief.

At the time of his admission to the clinic, examination disclosed the classical signs of aortic insufficiency with marked enlargement of the heart, emphysema, râles at the bases of the lungs, moderate enlargement of the liver and slight edema of the dependent parts, moderate dilatation of the arch of the aorta, peripheral arteriosclerosis, albuminuria and a slight elevation of blood pressure—

*S. H. No. 7589.

190/60 mm. of mercury. The pleuro-pulmonary changes, however, were of particular interest. The thorax was long, narrow and deep and the lungs in general were hyperresonant. Below and lateral from the angle of the right scapula there was a small round area of diminished resonance and here the breath sounds were more distant and the râles were more numerous and persistent than elsewhere.

In the absence of symptoms of infection, the atypical physical signs in the lower half of the right lung suggested the presence of a metastatic neoplasm and a roentgenograph of the chest made the following day showed two oval shadows at the site corresponding to the area of altered physical signs that confirmed that impression. (Figure 1.)

The results of a very complete survey including all of the usual investigations of the laboratory gave no other positive informative data. Although there were no extra-cardiac evidences of syphilis—iritis, perforation of the nasal septum, proliferative periostitis, orchitis or general adenopathy—and although the Wassermann reaction was negative, the patient was given 20 grains of potassium iodide thrice daily. Rest in bed, adequate doses of digitalis and the usual limitation of fluid, of food and of sodium chloride were followed by rapid symptomatic improvement. A roentgenograph of the chest made on December 27, 1929, demonstrated unexpectedly that the ovoid basal shadows had disappeared and this finding was confirmed in roentgenographs made on January 2, 9 and 23, 1930. (Figure 2.)

Potassium iodide was given from December 16, 1929, to January 24, 1930, and at that time the patient left the hospital with the instruction that he continue this medication.

He returned to the institution on February 10 and stated that for three weeks he had been asymptomatic in spite of the fact that he had been active physically and had taken no medicine, but that about February 4 he had a recurrence of paroxysmal dyspnea with unproductive cough. A re-examination showed that the signs of partial cardiac decompensation, present on December 8, had recurred. Again, the small area of dullness with suppression of breath sounds and râles that had disappeared before his discharge from the institution were detected and as before, ovoid shadows, the exact replicas of those seen previously, were shown in the roentgenographs made at

this time. (Figure 3.) Rest, digitalization, limitation of the intake of fluids, of solids and of salines, and the administration of 30 grains of potassium iodide every four hours were resumed on February 20, 1930, and in the films made on the 25th, when his symptoms had subsided once more, these shadows were no longer visible. (Figure 4.)

At this time the diagnosis of the pleuro-pulmonary condition was not clear. A neoplasm was excluded by the evolution of the lesion and by the absence of any source from which a metastasis could have come. A benign tumor could not have shown such rapid appearance and reappearance and an hydatid cyst—had one been present and ruptured—would have given rise surely to local or to constitutional symptoms. A mycotic infection would have had a different distribution and course, and an infarct would have been attended by local symptoms and a source for embolism would probably have been detected. Had the condition been due to a bronchial cyst that had evacuated its contents into a bronchus or into the pleura, enlightening manifestations should have been apparent. An encapsulated empyema was excluded by the absence of febrile and leucocytic reaction and by the disappearance and reappearance of the physical signs. Inasmuch as there had never been hemoptysis, expectoration, pleural pain or constitutional disturbances indicative of an intoxication, a basal tuberculoma was excluded, although there were signs of healed apical foci. Because of the fact that the abnormal findings seemed to be influenced by the administration and by the withdrawal of potassium iodide, the fallacy of *post hoc ergo propter hoc* was nearly committed and gumma of the lung considered. The rarity of that condition, the rapidity with which the area of increased density disappeared after the ingestion of small doses of iodide, the negative history and the negative Wassermann reaction, seemed to exclude syphilis as the primary cause of the condition. For a time, a lobular atelectasis secondary to a small gumma of a bronchus was considered, only to be excluded by the subsequent development of the case.

From March until May, 1930, the patient was able to be about with little discomfort so long as his physical activities were restricted. On May 12, he returned to the clinic with a recurrence of his symptoms and with the same physical signs.

On May 14, after a roentgenograph of the chest had been taken,

(Figure 5), an exploratory thoracentesis was done. A needle was introduced into the area of atypical signs in the right infrascapular region and 80 c.c. of slightly cloudy yellow fluid were aspirated. The fluid contained 90 cells per c.mm., 90% of which were lymphocytes and 7.5 gms. of albumin per liter. It was sterile bacteriologically and gave a negative Wassermann reaction. A roentgenograph made after this procedure showed an almost complete disappearance of the upper and more lateral of the oval shadows that were present just prior to the paracentesis. (Figures 6 and 7.) This finding established beyond peradventure the diagnosis of an encapsulated hydrothorax.

After his discharge from the hospital in an improved condition, the patient relapsed and was readmitted on October 12th, 1930. Once more, the ovoid shadows were present (Figure 8) and following a period of rest and digitalis, this time not supplemented by the administration of potassium iodide, the abnormal shadows disappeared within two weeks. (Figure 9.)

Another period of comparative well being followed and lasted until April 11, 1931, when he was admitted to the Johns Hopkins Hospital with a return of his symptoms of myocardial weakness and of anginoid pain and a diagnosis was made of arteriosclerosis, syphilis of the aorta, aortic insufficiency, myocardial insufficiency, pulmonary tuberculosis and pleurisy with encapsulated effusion.

An electrocardiograph showed left ventricular preponderance and a normal sino-auricular rhythm. There was slurring of the QRS complexes in all leads, with normal A-V and inter-ventricular conduction times. Although the T-wave was inverted in lead I, biphasic in leads II and III, and in all leads arose from the QRS complexes above the iso-electric line, these waves were not considered characteristic of coronary disease.

Rest and the therapy were without favorable effects. On May 8, fever, leucocytosis with signs of an effusion developed at the left base and moist râles were heard in the upper portion of the right lung. A thoracentesis yielded 100 c.c. of blood-tinged exudate (specific gravity 1020, 1040 cells per c.mm., of which 98% were polymorphonuclears and the albumin was 4 plus).

A week later, a second thoracentesis yielded 100 c.c. of bloody fluid with a specific gravity of 1019, containing 3850 cells per c.mm.

(75% P.M.N.'s) and 18 grams of albumin per liter. These fluids were sterile. There was albuminuria and occasional cylindruria. The hemotological findings were normal, except for a polymorphonuclear leucocytosis. The liver was moderately swollen.

On May 31, 1931, the patient died and a post-mortem examination was made.

SUMMARY OF AUTOPSY, JOHNS HOPKINS HOSPITAL, No. 12025.

"Syphilitic aortitis; aortic insufficiency; obliteration of orifice of right coronary. Scar in left ventricle. Cardiac hypertrophy and dilatation. Chronic passive congestion of liver, spleen and lungs. Old infarct of lung. Left hydrothorax. Healed infarct of left kidney. Bilateral apical scars and calcification. Pleural adhesions. Adentia."

The right lung was partially collapsed, and within the firm apex there were several calcified nodules, but otherwise it showed nothing significantly abnormal. The left lung, too, contained several calcified apical tubercles. Both lungs were emphysematous, and showed chronic passive congestion and an old infarct.

The condition of the pleura was of especial interest. On the right side, a single strong band of adhesions extended from the old tuberculous area at the apex of the lung to the parietal wall of the chest and the lower portion of the pleural cavity was obliterated by a number of small, firm adhesions. The pleural layers of the fissure between the lower and the middle lobes were fused completely, except in the anterior axillary line at the level of the eighth rib, where there was a cavity 3 cm. in diameter between them. Similar conditions were found posteriorly in the fissure between the upper and the middle lobes. The left thoracic cavity showed an old adhesive pleurisy also and there was a definite pocket containing fluid beneath the base of the lung. On the right side, there was no free fluid and only 100 c.c. of clear fluid were found on the left side. The location of the two pockets found in the interlobar fissure of the right lung corresponded to the site of the ovoid shadows seen in the roentgenographs of the chest and to that from which fluid was aspirated during the life of the patient.

The heart was enlarged greatly, due in the main to an aortic insufficiency and an enormous increase in the size of the left ventricle. Near the apex of that chamber and in a small area of the

inter-ventricular septum near it there was a well-defined depression covered by opaque, thickened endocardium. The papillary muscles about this region were atrophied and the myocardium there was less than half of the average thickness of the ventricular wall. The lesion was obviously the site of an old infarct. The orifices of the coronary arteries were narrowed markedly—that of the right one was practically obliterated, but no obstruction was found within any of the coronary vessels. The aorta showed a thickening of the intima, yellow and partially calcified, extending from the sinus of Valsalva to the junction of the thoracic and the abdominal portions of the vessel, where the abnormal appearance ended abruptly. Just proximal to the termination of this thickening, a few linear striations were seen typical of syphilitic aortitis. At the beginning of the descending arch there was an aneurysmal-like pouching of the vessel.

The anatomical findings confirmed the ultimate clinical diagnosis. They proved that the loculation of the serous transudate that was formed during the periods of congestive myocardial failure resulted because an antecedent obliterative pleurisy, doubtless of tuberculous origin, had left only interlobar lacunae in which the fluid could collect. Why there was no fluid free in those other portions of the right thoracic cavity in which the parietal and visceral layers of the pleura were not adherent lacks a satisfactory explanation. The small encapsulation of fluid at the base of the left thorax, not detected before the post-mortem examination, was due probably to a recent tuberculous pleurisy, for the fluid demonstrated there contained numerous leucocytes, much albumin and had the other cardinal characteristics of an inflammatory exudate.

It is worthy of comment that all of the cases of encapsulated hydrothorax recorded in cases of circulatory failure have occurred in patients with disease of the coronary arteries or of the aortic area near the ostia of these vessels, but for this no explanation is apparent. Perhaps it is a coincidence only. That the sacculations have all been located on the right side is concordant with the relative frequency of right hydrothorax generally in cases of cardiac decompensation.

Inasmuch as adhesive pleurisy occurs in many individuals and as myocardial failure with the development of hydrothorax is a frequent clinical event, it may be that the two pathological states are

coexistent more frequently than reported instances indicate. Perhaps if the coincidence were borne in mind and sought for, hydrothorax saccatus would be found to occur with some regularity in the type of patient considered. To determine whether or not this idea is valid, it seems desirable to examine roentgenographically all patients with hydrothorax before and after a thoracentesis and to project the X-ray sagittally and laterally as well as antero-posteriorly. In addition, whenever the thoracic roentgenograph of a patient with cardiac disease shows a shadow that indicates the presence of encapsulated fluid an exploratory thoracentesis should be done.

The records of the patients discussed illustrate the truth of the aphoristic statement that queer cases are usually abnormal types of common conditions.

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DISCUSSION.

DR. CLEAVELAND FLOYD: Not infrequently in the routine X-ray of cases, looking for tuberculosis, one comes across a clear chest plate with only the presence of interlobular thickening running out well toward the periphery of the lobar space and more commonly, I think, on the right than on any other situation. What I wanted to do was to ask Dr. Austrian if these cases which one runs across occasionally, with no other signs in the case, are in all probability an old tubercular infection between the lobes or, as in cases such as he cites, of hydrothorax complicated with heart condition. In the cases that I think of there were no clinical evidences of heart lesions.

DR. HUGH M. KINGHORN: Two very important lessons, I think, have come out of this paper: One is that where you have a localized hydrothorax you know you ought to always put in a needle.

I have had a case recently of a far advanced tuberculosis of the lungs who had also diabetes, serious diabetes, who developed a localized pneumothorax. There was no evidence of fluid that could be seen by the X-ray findings. The surprising point to me was that, although this man was going down-hill, I thought it was from his diabetes; the pulmonary condition seemed to improve. The pulmonary condition was beneath the localized pneumothorax. The local-

ized pneumothorax was situated over the right base behind and extended for about an inch or an inch and a half in area. It was a surprising thing that the physical signs improved constantly, I thought probably due to the localized pneumothorax. There was no evidence of fluid whatever. At autopsy, however, there was about from 10 to 20 cc. of a foul-smelling pus. I made the mistake that I did not put in a needle.

I think it is very, very necessary to do this, and that was the great lesson, that I should have put in a needle in spite of the fact that no fluid level was visible. You might say that if I had gone too far perhaps I might have injured the lung. That is quite a fact. At the same time, if I had recovered the fluid I might have done him some good because the lung had ruptured, but it was a closed cavity, so to speak; it did not connect with the bronchus. So the important lesson to me in this case was, from Dr. Austrian's case, where you have a localized pneumothorax, put in a needle and see if you can withdraw pus in spite of the fact that you may not have fluid.

DR. AUSTRIAN: In reply to Dr. Floyd's question, may I say I think it is generally accepted that these delicate bands, apparently referable to the thinning of the pleura, are an evidence of an old pleurisy with thickening along the fissure. These can be demonstrated to be such, particularly if one takes these lateral or angular views of the chest.

With reference to Dr. Kinghorn's remarks, I may say this: that, of course, what we have demonstrated roentgenographically of local encapsulated fluid is time-worn and old. The only particular interest is, it seems to me, that from that patient's standpoint we are dealing with a transudate that is walled off in this fashion, a point of view that one does not usually consider—at least we have not—when one sees shadows of that type. It raises, as an interesting matter for consideration and diagnosis, the interpretation of abnormal pleural pulmonary findings in a patient with partial cardiac degeneration, and the fact needs always to be emphasized that one may not have a complicating state of affairs but simply a usual happening with an unusual localization.